can be given. As sputum cultures are notoriously unreliable in this regard, other techniques, including transtracheal aspiration, collecting bronchoscopic material with protected catheters, transthoracic needle aspirates and cultures of empyema fluid (when present) are often indicated in compromised hosts or inpatients.

Prolonged high-dose penicillin therapy is still the standard therapy for community-acquired lung abscesses, which are usually due to anaerobic organisms. Recent reports, however, of penicillin-resistant anaerobic organisms and treatment failures with penicillin administration have led to suggestions that clindamycin may well be the antibiotic of choice, particularly if *Bacteroides* species is identified, if penicillin allergy is suspected, if a patient fails to respond to penicillin therapy and perhaps if a patient is seriously ill without any of the aforementioned factors being present. Chest physiotherapy may be beneficial in many cases as an adjunct to antibiotic therapy, and the use of bronchoscopy to rule out an organic endobronchial obstruction and to improve drainage is still advocated by many, although probably not required as a routine measure. Abscess drainage may be improved by passing a balloon-tipped catheter through the bronchoscope to permit lavage of the affected area.

Surgical excision of chronic abscess cavities, commonly done in the preantibiotic era, is now rarely indicated. Recent reports of successful percutaneous drainage, either by pneumonotomy or tube thoracostomy, or of chronic lung abscesses that have failed to close after appropriate antibiotic therapy, suggest that such modalities may play an increasingly important role, particularly in critically ill patients.

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Air Travel and Patients With Pulmonary Problems

FLYING ABOARD COMMERCIAL jet aircraft exposes passengers to many environmental stresses, including altitude-induced hypoxia and decreased atmospheric pressure. These stresses are ameliorated by cabin pressurization (to between 5,000 and 8,000 ft) and are well tolerated by most persons, including those with mild cardiorespiratory disorders. Patients with more severe chronic pulmonary conditions and hypoxemia (arterial oxygen tension [Pa_{O_2}] of less than 70 mm of mercury) at ground level, however, are potentially vulnerable to significant hypoxia-related cardiopulmonary or neurologic (or both) dysfunction and symptoms even at moderately high altitudes. In these persons the risk of clinical sequelae is increased by smoking (with increased carboxyhemoglobinemia), alcohol ingestion, exercise, sleep and the drying effects of the cabin environment on respiratory secretions.

Preflight evaluation of patients with chronic lung disease should rely on both objective test results and clinical judgment. Patients with resting or exertional dyspnea, cyanosis, cor pulmonale, bullous lung disease, a vital capacity of less than 50% of predicted, a maximum voluntary ventilation of less than 40 liters per minute, hypercapnia or a Pa_{O_2} (measured while a patient is breathing room air) of less than 50 mm of mercury are considered at high risk for altitude-related morbidity. An advising physician must also consider coexisting anemia, hemoglobinopathy and disorders of the cardiovascular and central nervous systems that may be exacerbated in a hypoxic environment. The findings of a routine history, physical examination, hemogram, serum electrolytes, electrocardiogram, chest radiograph, spirometry and exercise tests may indicate serious abnormalities (at ground level) but these tests are limited in predicting altitude tolerance or Pa_{O_2}. The most practical and accurate discriminator of altitude Pa_{O_2} is the Pa_{O_2} at ground level. A formula has been developed for predicting altitude Pa_{O_2} in normocapnic patients with chronic obstructive pulmonary disease at elevations between 5,000 and 10,000 ft:

$$\text{altitude } Pa_{O_2} = 22.8 - 2.74x + 0.68y$$

where x = anticipated (cabin) altitude in thousands of feet and y = room-air resting Pa_{O_2} in mm of mercury at ground level. Useful information regarding symptomatic, cardiovascular and neuropsychological responses to acute hypoxia (the major threat of altitude) may be obtained directly with a hypoxia-altitude simulation test that may be done in many pulmonary function laboratories. This stress test involves the breathing of hypoxic gas mixtures that simulate the inspired oxygen concentration of one or more anticipated altitudes and the monitoring of arterial oxygenation (by oximetry or an indwelling arterial catheter), electrocardiogram and symptoms, with or without light exercise or supplemental oxygen. An altitude Pa_{O_2} of less than 50 mm of mercury, oxygen saturation of less than 85% (or higher thresholds, depending on clinical judgment) or the presence of adverse cardiac or other responses signal the need for possibly corrective intervention, such as supplemental oxygen, drug therapy or smoking cessation, or advice to forego air travel.

A patient with pulmonary problems who is considered a stable candidate for commercial air travel may require further assistance from his or her physician, oxygen vendor and airline(s), depending on the patient's fitness and respiratory status, need for supplemental oxygen and flight itinerary. The physician may be asked to prescribe oxygen requirements and other special needs. The oxygen vendor may need to provide services at stopovers, the final destination and for the return flight. American carriers cannot legally allow passengers to use their personal oxygen units aboard aircraft. Most major airlines in the United States will provide inflight oxygen with advance notice. Unfortunately, airline policies differ widely regarding oxygen capabilities and inflight use of respiratory equipment. These issues, if applicable, need to be inquired about in detail. Further advice and assistance can be obtained from local pulmonary rehabilitation programs and the California Thoracic Society, American Lung Association of California, 424 Pendleton Way, Oakland, CA 94621.

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Noninvasive Determinations of Arterial Oxygenation and CO_2 Tension

MOST ARTERIAL BLOOD analysis is done to evaluate oxygenation (partial pressure of arterial oxygen [Pao_2]) and adequacy of ventilation (arterial carbon dioxide tension [$Paco_2$]). It is now technically possible to do these measurements noninvasively. Noninvasive determination of arterial O_2 saturation by transmitting two infrared wavelengths through the ear capillaries was developed in 1935 but was clinically unacceptable because of difficulties in calibration and inaccuracy in the clinical setting. Improved technology in the 1970s, using two to eight wavelengths, coupled with analysis of the pulse of arterial blood in the ear heated to 39°C, allowed for simplified calibration and clinically useful ear oximetry. Simultaneously, neonatologists found that the blood O_2 electrode applied to the skin heated to 44°C could give accurate estimates of Pao_2 when blood flow to the skin was adequate. Such measurements have largely replaced arterial blood gas analysis in neonatal intensive care units. In adults, the thickened skin (physiologic if not sociologic) made transcutaneous O_2 tension ($Ptco_2$) less reliable.

Similarly, the transcutaneous CO_2 pressure can be measured using either infrared transmission or the blood CO_2 electrode. Accuracy still depends on adequate blood flow to skin but less so than $Ptco_2$ since a continuous flux of CO_2 through the skin is not needed. CO_2 can also be measured in the exhaled air by infrared analyzers or mass spectrometry. The end-tidal CO_2 pressure ($Petco_2$) approximates $Paco_2$, and rebreathing methods can give better approximation of the mixed venous CO_2 pressure ($Pvco_2$).

These noninvasive methods are being increasingly applied clinically in the 1980s. As technology improves and becomes more familiar, their use can be expected to increase and replace arterial blood gas determinations in adult patients, as it already has in neonates. Several well-established applications for ear oximetry are as follows: to monitor for sleep apnea and hypoxemia during sleep studies; to adjust the amount of inspired O_2 at the bedside to achieve the desired O_2 saturation (usually more than 90% or a Pao_2 of more than 60 torr); to monitor oxygenation during exercise studies, and to monitor oxygenation in critically ill patients in intensive care units. These measurements should be verified by an initial simultaneous arterial blood analysis, particularly in a critical care setting or when the data are not consistent with the clinical assessment. Repeated arterial punctures to monitor oxygenation, however, are clearly no longer necessary or desirable.

Transcutaneous O_2 monitoring may also be used in the above settings but is more time-consuming to set up, requires higher temperatures (44°C), leaving a burn on the skin and therefore needs to be moved every three to four hours, and is more critically dependent on skin blood flow for accuracy.

Noninvasive monitoring of CO_2 is less common and usually applied to critically ill patients where ventilatory failure is imminent or present and endotracheal intubation and mechanical ventilation are indicated. A single skin electrode is now available to monitor both $Ptco_2$ and $Ptcco_2$. A single infrared unit can be used to alternately monitor both airway CO_2 ($Petco_2$) and $Ptcco_2$. The $Petco_2$ underestimates the $Paco_2$, and $Ptcco_2$ tends to overestimate the $Paco_2$. Monitoring both $Petco_2$ and $Ptcco_2$ can provide a bracket to estimate $Paco_2$. Changes in $Petco_2$ or $Ptcco_2$ indicate either changes in $Paco_2$ or in the gradient between $Paco_2$ and $Petco_2$. The $Paco_2$-$Petco_2$ gradient increases with abnormalities in ventilation (low tidal volume or maldistribution of ventilation), while the $Paco_2$-$Ptcco_2$ gradients increase when skin blood flow decreases or increases $Pvco_2$ due to metabolic changes. Therefore, a change in a noninvasive CO_2 measurement indicates either true $Paco_2$ changes, not uncommon in mechanically ventilated patients, or possibly a clinically significant pathologic change in ventilation, blood flow or metabolism. "Smart" alarm systems capable of analyzing such complex physiologic data to differentiate artifact from actual changes are technically possible, but the utility or necessity in a patient with critical ventilatory and circulatory problems is yet to be determined. For now, successful use of noninvasive CO_2 measurement depends on a "smart" and experienced critical care team.

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Home Oxygen Therapy

HOME OXYGEN THERAPY is indicated to correct hypoxemia and prevent the adverse cellular effects of hypoxia reflected in abnormalities of brain, heart and lung function. Hypoxemia is assessed by clinically evaluating a patient and by directly measuring arterial blood oxygen tension and saturation.

The management of hypoxemia due to chronic lung disease is the principal clinical indication for home oxygen therapy. Ongoing oxygen therapy should be prescribed for those patients who, after a month of optimal medical management, show a resting, nonrecumbent arterial oxygen tension of less than 55 mm of mercury or an arterial oxygen saturation of less than 85%. Patients with evidence of pulmonary hypertension, impaired mentation or erythocytosis qualify for home oxygen therapy if their arterial oxygen tension is less than 60 mm of mercury.

The National Institutes of Health Nocturnal Oxygen Therapy Trial showed the superiority of continuous over nocturnal oxygen therapy in the treatment of hypoxemia due to chronic obstructive pulmonary disease.

When hypoxemia occurs only during sleep, or when supine, and if the arterial oxygen tension falls to less than 55 mm of mercury or the oxygen saturation to less than 85%, oxygen therapy during sleep, or when supine, is indicated.